PRIMARY RAYNAUD'S DISEASE IN A YOUNG ADULT: A CASE REPORT

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ABSTRACT

Raynaud's phenomenon (RP) is characterized by intermittent vasospasm and subsequent ischemia of the extremities, typically triggered by cold exposure or emotional stress. This leads to a distinct triphasic discoloration of the fingers or toes, progressing from pallor to hyperemia. This case report describes a 20-year-old woman presenting with a 1-year history of episodic bilateral finger discoloration (pallor, cyanosis, erythema) triggered by cold exposure, consistent with primary Raynaud's phenomenon. Physical examination revealed no digital ulcers or skin changes, and basic laboratory tests were unremarkable, though autoimmune serology was unavailable due to resource limitations. The patient's photograph documenting an acute attack provided valuable diagnostic support. Management emphasized conservative measures including cold avoidance and behavioral modifications, which successfully controlled symptoms. This case highlights the clinical diagnosis of primary RP in resource-constrained settings, the utility of patient-documented evidence, and the effectiveness of non-pharmacological approaches.

Keywords: Ischemia, Raynaud phenomenon, Vasospasm.

INTRODUCTION

Raynaud's phenomenon is characterized by intermittent vasospasm and subsequent ischemia of the extremities, typically triggered by cold exposure or emotional stress. This leads to a distinct triphasic discoloration of the fingers or toes, progressing from pallor (white) to cyanosis (blue) and finally to reactive hyperemia (red). The condition can be classified as primary (idiopathic, occurring without an underlying disorder) or secondary (associated with diseases such as autoimmune disorders)(Goundry et al., 2012).

Epidemiological studies suggest that Raynaud's phenomenon affects approximately 5–20% of women and 4–14% of men in the general population, with a lower prevalence (0.1–1%) among individuals aged 60 and older. Primary Raynaud's, often benign and more prevalent in young women aged 15–30, it may also exhibit a familial predisposition (Temprano, 2016).

Secondary Raynaud's is frequently linked to systemic conditions, particularly systemic sclerosis and mixed connective tissue diseases. Literature reported that at least 12.5% of patients with Raynaud's phenomenon progress to scleroderma, and 13.6% develop

connective tissue disorders (Garner et al., 2015; Goundry et al., 2012).

This case report examines a young patient with Raynaud's disease, focusing on the diagnostic evaluation of this condition.

LITERATURE REVIEW

Definition

Raynaud Phenomenon (RP) is a transient, recurrent vasospastic disorder affecting peripheral blood vessels, marked by reversible episodes of digital ischemia followed by reperfusion. Clinically, this manifests as sequential color changes in the affected digits: initial pallor (white) due to arterial constriction and ischemia, progressing to cyanosis (blue) from tissue hypoxia, and finally erythema (red) resulting from reactive hyperemia upon vasodilation (Herrick, 2017).

RP may represent either an exaggerated physiological response to cold exposure or a secondary manifestation of an underlying systemic disease. In some cases, it can also serve as a critical indicator of severe digital ischemia requiring prompt clinical evaluation (Nawaz et al., 2022).

Pathophysiology

RP develops through three key pathophysiological processes: diminished blood flow, vascular constriction, and dysregulated neurogenic and inflammatory responses. The somatosensory system plays a crucial role in temperature perception, where cold exposure activates Aδ and unmyelinated C-fibers, ultimately stimulating TRPM8 cold receptors (Musa & Qurie, 2025).

These receptors mediate cutaneous vasoconstriction, thermoregulatory responses, and cold avoidance behaviors. During cold exposure, sympathetic activation induces the release of vasoconstrictive neuropeptides and norepinephrine, reducing cutaneous perfusion. In secondary RP, endothelial dysfunction further exacerbates vasoconstriction through endothelin-1 secretion (Musa & Qurie, 2025).

Primary RP involves heightened α2-adrenergic receptor sensitivity in digital vessels, amplifying vasoconstrictive responses to cold and emotional stress. These receptors, concentrated in distal arterial smooth muscle, are modulated by sympathetic input. Clinical evidence shows that α2-adrenergic blockade can mitigate cold-induced vasospastic episodes. In contrast, secondary RP reflects underlying vascular pathology, where diseases like systemic sclerosis cause endothelial damage, vascular fibrosis, and impaired vasoreactivity, ultimately leading to tissue ischemia. This distinction underscores the importance of differentiating primary from secondary RP in clinical evaluation (Musa & Qurie, 2025).

Clinical Presentation

RP manifests through distinctive vascular color changes in affected extremities, most commonly presenting as a triphasic transition from pallor (ischemia phase) to cyanosis (hypoxia phase) and finally erythema (reperfusion phase). However, this classic progression may be incomplete in some cases, with patients exhibiting only pallor, cyanosis, or a biphasic pattern ending with reactive hyperemia. The distribution of involvement varies significantly, while some cases affect a single digit, others involve multiple fingers or toes. Notably, thumb involvement often indicates secondary RP and

warrants investigation for underlying pathology (Choi & Henkin, 2021).

Clinically, RP episodes are frequently accompanied by ischemic pain due to compromised sensory nerve perfusion. Characteristically, symptoms occur in discrete, self-limited episodes triggered by cold exposure or emotional stress, with complete resolution between attacks. The manifestations typically demonstrate bilateral symmetry in both upper and lower extremities. Although symptom duration can range from minutes to hours, most episodes resolve within 15-20 minutes following rewarming. This temporal pattern, along with the characteristic color changes, helps distinguish RP from other vascular disorders (Pauling & Matucci-Cerinic, 2024).

Diagnosis

The diagnostic evaluation of RP does not include provocative cold testing as a recommended diagnostic approach. Instead, established diagnostic criteria have been developed to classify the probability of RP based on clinical presentation (Pauling & Matucci-Cerinic, 2024):

1. Definite RP: Repeated episodes of biphasic color changes upon exposure to cold.
2. Possible RP: Uniphasic color changes plus numbness or paresthesia upon exposure to cold.
3. No RP: No color changes upon exposure to cold.

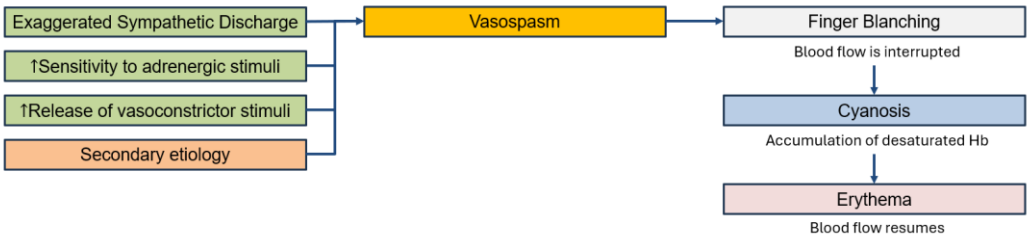

Figure 1. Pathophysiology of Raynaud Disease

Management

As the drug-based treatment of RP is still being investigated, general and lifestyle modifications are usually the first steps in management. Symptoms of RP can be controlled by triggers avoidance that can elicit a vasospastic attack. Avoiding the cold and keeping the body warm through thick clothing, gloves, orpadding can prove useful. Alleviation of emotional stress and anxiety can also decrease the frequency of the onset of attacks in some groups of patients (Kwakkenbos et al., 2024).

RESEARCH RESULT

Case Presentation

A 20-year-old woman presented with episodic bluish-to-white discoloration of her fingers upon exposure to cold temperatures, persisting for the past year. The symptoms affected all fingers bilaterally and were accompanied by numbness and pain. These episodes occurred 2–3 times per week, typically triggered by cold air or water contact. There was no family history of similar symptoms or underlying systemic illnesses.

Physical examination revealed no digital ulcers, skin thickening, or signs of connective tissue disease. Based on the classic triad of pallor,

cyanosis, and erythema in response to cold she was initially diagnosed with Raynaud's disease, but patient brought a picture she captured in times of exacerbation (figure 2). To further confirm the possibility of secondary etiology of Raynaud's disease, patient was referred to local hospital and comprehensive laboratory tests (including complete blood count, inflammatory markers, and renal function) yielded normal results (table 1). Autoimmune serology (e.g., antinuclear antibodies) could not be performed due to limited resources.

Based on the clinical presentation and exclusion of secondary causes, a diagnosis of primary Raynaud's phenomenon was confirmed. Conservative management was initiated, emphasizing cold avoidance, behavioral modifications (e.g., wearing gloves), and regular follow-up to monitor for potential progression or development of autoimmune features. The patient was advised to seek prompt re-evaluation if symptoms increased in frequency or severity.

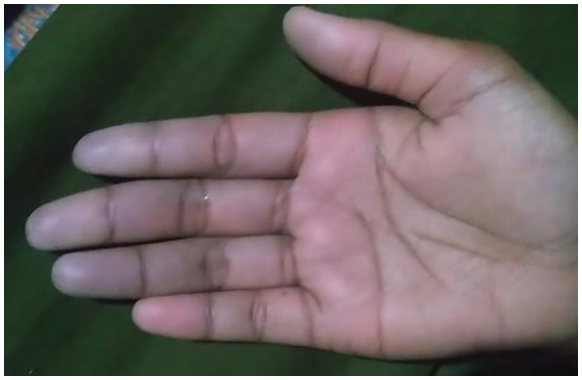

Figure 2. Patients Documented Finger in Times of Exacerbation

Table 1. Laboratory Findings

Index	Value
Hemoglobin	13.4 g/dL
Hematocrit	39.8%
Leukocyte	8.08 x 10^3/mm^3
Thrombocyte	298 x 10^3/ mm^3
Rheumatoid Factor	11 IU/mL
D-Dimer	350 ng/mL
PT	19.4 s
INR	1.39 s
APTT	60.0

DISCUSSION

This case report describes a 20-year-old woman presenting with classic symptoms of primary Raynaud's phenomenon (RP), including recurrent, cold-induced triphasic color changes (pallor,

cyanosis, and erythema) in all fingers bilaterally, accompanied by pain and numbness. The absence of digital ulcers, skin thickening, or systemic symptoms, along with normal basic laboratory findings, strongly supports the diagnosis of primary RP. This clinical presentation aligns well with established diagnostic criteria for primary RP, which emphasize episodic vasospastic attacks triggered by cold or stress without evidence of underlying connective tissue disease. The patient's young age and female gender are also consistent with known epidemiological patterns of primary RP, which predominantly affects women aged 15-30 years (Garner et al., 2015; Orsini et al., 2021).

However, the inability to perform autoimmune serology testing (particularly antinuclear antibodies) due to resource limitations presents an important consideration. While the clinical presentation strongly suggests primary RP, current literature recommend excluding secondary causes through comprehensive evaluation, including autoimmune markers.

This case highlights the diagnostic challenges that may arise in resource-limited settings, where clinicians must rely more heavily on clinical judgment. Interestingly, the patient's photograph documenting an acute episode (Figure 2) provided valuable objective evidence supporting the diagnosis, demonstrating how patient-reported outcomes can supplement clinical assessment when advanced testing is unavailable.

The management approach in this case focus on conservative measures like cold avoidance and behavioral modifications is entirely consistent with current treatment recommendations for primary RP. The patient's good response to non-pharmacological interventions reinforces existing evidence that lifestyle modifications alone are often sufficient for mild cases.

However, the recommendation for regular follow-up is particularly important, as approximately 10-20% of patients initially diagnosed with primary RP may eventually develop signs of connective tissue diseases. This potential for progression underscores the need for ongoing monitoring, even in seemingly straightforward cases. The case also illustrates the importance of patient education in RP management, as understanding trigger avoidance can significantly improve quality of life for these patients (Garner et al., 2015).

CONCLUSION

This case demonstrates classic primary Raynaud's phenomenon diagnosed clinically in a resource-constrained setting, emphasizing that thorough history and examination can reliably identify typical cases. While behavioral interventions provided adequate symptom control, the necessity of monitoring for disease progression underscores the dynamic nature of RP. The case validates current diagnostic algorithms and treatment hierarchies while highlighting real-world diagnostic challenges.

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